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LETTERS TO THE EDITOR

The Educational Role of Thrombolytic Therapy

The recent article by Gorlin (1) on the present status of thrombolytic therapy in acute myocardial infarction needs further comments. I agree wholeheartedly with Gorlin when he concludes that "a small overall gain has been made with thrombolysis, which can benefit a small subset of patients with acute myocardial infarction, provided that both the immediate and the long-term risks are carefully assessed." In most instances thrombolytic therapy is failure-expensive medicine. If one accepts the laboratory data that 85% of heart muscle death takes place within the first 2 h, it appears to be almost impossible to give effective thrombolytic therapy within that short time range. The average time the patient has prolonged chest pain before hospital arrival is approximately 1.5 h. In the emergency room, there is still significant delay [mean about 1.5 h (2)]. Even when thrombolytic therapy is given, there is another 45 min before the vessel is open. Therefore, most patients appear to get the drug too late.

What Gorlin does not take into account is that the benefits of thrombolytic therapy may not be in the treatment of patients with myocardial infarction but in the publicity given to early care of the patient with ischemic disease. The awareness program for early intervention will bring into the hospital setting many patients with unstable angina, and this is what will be most important in the long run.

The Paul Dudley White Coronary Care System at Saint Agnes Hospital (3) has had a chest pain emergency room for the last 8 years and has provided early cardiac care with an educational program aggressively given within its community. This early cardiac care approach has increased the number of admissions from 1,000 to 2,000 during this period of time and has changed the subsets so that the proportion of patients with unstable angina is now within the 60% range.

The emphasis on early cardiac care may help reduce the time frames for treatment of acute myocardial infarction, but in most cases it will be expensive medicine given too late. Perhaps paramedic administration of therapy or fast tracks by medical residents within hospitals may improve this picture, but in most cases significant damage has been done. The "Golden Fleece," namely the patient with unstable angina, may be a by-product of this enthusiasm for thrombolytic therapy and may provide us with an enhanced delivery system for patients seen before the occurrence of cardiac damage and sudden death. In this setting, an educational program for chest pain awareness similar to the present programs for hypertension, cigarette smoking and cholesterol could provide us with an excellent tool for significantly reducing mortality and morbidity. To do so, all 6,700 hospitals in the United States must provide this link-up between the community and early cardiac care.

It is my viewpoint that Gorlin (1) may have missed the mark by not appreciating a more panoramic view of what thrombolytic therapy can mean in the long run.

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Reply

I appreciate very much the comments expressed by Bahr reminding all of us that patients must get to hospital sooner if improvements are to be made in the rate of survival. Thrombolytic therapy is only one of a number of proposals for early therapy that have been made over the years. Bahr may recall that Pantridge and Geddes (1) in Belfast directed attention to the number of patients who might be salvageable if seen within *minutes* of the inception of a myocardial infarction. So it was that Sarnoff (2) proposed the development of early electrocardiographic alert systems, including prepackaged antidotes for arrhythmias. So it was that the community-wide Nottingham study (3) tested the difference in the outcome of patients rushed to the hospital versus those treated at home by physicians. Thus the concept of seeing the patient earlier and earlier, at a point where one can intervene appropriately, is not new and is as unlikely to be a by-product of thrombolytic therapy any more than it was of antiarrhythmic and defibrillating therapy in an earlier generation.

Bahr makes a good point in that the public needs to be educated, but education is only the first step. Aspects of patient behavior such as denial, nonspecific symptomatology and the confounding component of hyporesponsiveness of patients to their complaints all play a part in delaying therapy.

It is worth examining why we are seeing more patients with so-called unstable angina than frank Q wave myocardial infarction. This trend predates thrombolytic therapy. It almost suggests that an alteration has occurred in the atheromatous plaque and its depth or extent or rupture, leading to a less catastrophic or complete arterial

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occlusion. This would affect the nature of myocardial ischemic insult and therefore the clinical presentation.

I thank Bahr for raising these very interesting questions.

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3. Rowley JM, Hampton JR, Mitchell JRA. Home care for patients with suspected myocardial infarction: use made by general practitioners of a hospital team for initial management. Br Med J 1984;289:403–6. LETTERS TO THE EDITOR

Color Doppler Diagnosis of Left Ventricular Pseudoaneurysm

Simultaneous with the report of Roelandt et al. (1), we described the two-dimensional Doppler color flow mapping and color-guided continuous wave Doppler features of a left ventricular pseudoaneurysm (2). Similarly, color Doppler demonstration of flow through a communication suspected by two-dimensional echocardiography was diagnostic of pseudoaneurysm. Color-guided continuous wave Doppler examination in the three cases of Roelandt et al. (1) revealed a "characteristic flow pattern" across the communication. They observed flow into the pseudoaneurysm during presystole and early to mid systole with an early systolic peak velocity (1.6 to 1.9 and 2.16 m/s, respectively) and reversed flow in late systole and early to mid diastole with an early diastolic peak velocity (1.7 and 1.27 m/s, respectively). By contrast, we observed holosystolic flow into the pseudoaneurysm with an early systolic peak velocity of 4 m/s and reversed holodiastolic flow with an early diastolic peak velocity of 2.2 m/s. Restricted left atrial filling due to compression by a huge pseudoaneurysm in our case, and differences in ventricular function, communication size and location and pseudoaneurysm compliance, may explain the different flow velocity patterns. Characterization of flow patterns across the communication requires further observations.

The cases of Roelandt et al. (1) and our own illustrate the advantages of Doppler color flow imaging. Doppler color flow imaging superimposes color-encoded flow signals on two-dimensional echocardiographic images in near real time. Simultaneous imaging of cardiac structure and spatially oriented blood flow allows rapid identification of a ventricular wall communication, detection of small and multiple communications and accurate delineation of communication size and concomitant flow abnormalities. Doppler color flow imaging enhances the accuracy, efficiency and confidence of two-dimensional echocardiography in diagnosing left ventricular pseudoaneurysm and facilitates recognition of this disorder.

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Reply

We thank Natello and Nanda for their comments regarding our report on the diagnosis of left ventricular pseudoaneurysm with use of Doppler color flow imaging and integrated continuous wave Doppler studies. We have also read their report (their Ref. 2) with interest. We are in total agreement with their comments on the value of Doppler color flow imaging in the diagnosis of left ventricular pseudoaneurysm.

The one area of divergence between the two reports is the characterization, by continuous wave Doppler study, of the velocity flow profile at the neck of the pseudoaneurysm. We accept that differences in the position of the pseudoaneurysm and differences in left atrial and left ventricular function may affect the pattern of flow between the left ventricle and the pseudoaneurysm.

We would recommend that future studies on the Doppler velocity flow profiles in left ventricular pseudoaneurysm should be directed by Doppler color flow imaging and include both pulsed and continuous wave Doppler examinations.

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